Wide complex tachycardia: differentiating ventricular tachycardia from supraventricular tachycardia

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INTRODUCTION

A wide QRS complex tachycardia (WCT) is defined as a tachycardia with QRS duration of >120 ms. Distinguishing between the different potential causes of a WCT can have important implications, particularly in terms of determining the urgency of treatment, deciding between different antiarrhythmic drugs and risk stratification for sudden cardiac death. Potential differential diagnoses for a WCT include ventricular tachycardia (VT), supraventricular tachycardia (SVT) with aberrant conduction, SVT with antegrade conduction via an accessory pathway (AP), ventricular pacing and ECG artefact (box 1). In most cases, paced ventricular activation and ECG artefact can be excluded with relative ease based on the ECG features and background clinical history. On the other hand, distinguishing VT from SVT may represent a challenge. In this regard, sufficient knowledge of the background history and detailed analysis of the ECG are central to making an accurate diagnosis.[1 2] This article will outline the approaches and algorithms for distinguishing between different forms of WCT with a particular focus on ECG characteristics to distinguishing VT from SVT.

CARDIAC ACTIVATION DURING VT AND SVT

In general, there are two major differences in the activation patterns that can be exploited when distinguishing VT from SVT: (1) the relationship between atrial and ventricular activation, and (2) the sequence of ventricular activation.

Relationship between atrium and ventricle

During SVT, the tachycardia originates from the atria or involves the atria in the tachycardia circuit. During VT, cardiac activation originates from the ventricle and atrial activation may or may not be linked to ventricular activation. In the event of VT with no VA conduction, the ventricle and atrium are dissociated. The presence of a less than 1:1 ratio of P wave to QRS complex (more 'vs' than 'As') is strongly suggestive of VT, with a specificity approaching 100%.[3 4] In contrast, an equal P–QRS relationship (1:1 ratio) does not reliably exclude VT as this pattern may occur due to retrograde VA conduction during VT. Of note, identification of AV dissociation on 12-lead ECG may be the only non-invasive means to distinguish VT and SVT due to antegrade AP conduction.

In addition to the relationship between P waves and QRS complexes, 'capture' and 'fusion' beats may also be used as markers of AV dissociation. Capture beats are characterised by a sudden narrowing of the QRS complex (*resembling the patient's baseline rhythm*) during WCT caused by ventricular activation entirely from the atria and using the specialised conduction system (figure 1). Fusion beats are characterised by a 'hybrid' QRS complex caused by collision of simultaneous ventricular activation from both ventricular and atrial sources. Importantly, differentiation between capture and fusion beats rely on a comparison with baseline ECG.

Learning objectives

- Review the underlying principles of physiological cardiac activation.
- Assess the differences in cardiac activation between ventricular and supraventricular tachycardias, and how these may relate to electrocardiographic changes.
- Discuss the electrocardiographic algorithms for diagnosis of wide complex tachycardia.

Patterns of ventricular activation

SVT with aberrant conduction activates the ventricle via the AV node with subsequent recruitment of all or part of the specialised His-Purkinje system. The rapid initial His-Purkinje–medicated activation is typically followed by a late phase of slower myocyte-to-myocyte activation, resulting in a typical RBBB or LBBB morphology (figure 2). VT on the other hand may originate from anywhere within the ventricle and is commonly associated with slow initial myocyte-to-myocyte conduction that may eventually engage the specialised His-Purkinje system. The differences in ventricular activation between SVT with aberrant conduction and VT result in 'typical' and 'atypical' QRS morphologies (table 1; figure 3). There are a number of specific features that differentiate between VT and SVT, including the QRS duration, QRS axis and QRS morphology. The specific features are discussed in more detail in the next section.

QRS morphology

An atypical QRS morphology, often assessed using leads V1/2 and V6, favours a diagnosis of VT (figure 3).[5] In LBBB-like tachycardia, atypical features include an rS complex with a prolonged r wave (>30 ms), a notched downsloping S wave, slow initial conduction (onset to nadir S wave of >60 ms), all in lead V1 or V2, or a q wave in lead V6.[6] In RBBB-like tachycardia, atypical features include a monophasic or biphasic QRS complex in lead V1, notched QRS with taller R (over R′) peak in lead V1[7] or R/S amplitude ratio <1 in lead V6.[3] A Q wave, either QR or QS pattern, in lead

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Box 1 Differential diagnosis of wide complex tachycardia

Causes of wide complex tachycardia
- Ventricular tachycardia
- Supraventricular tachycardia with aberrancy
- Supraventricular tachycardia with antegrade accessory pathway conduction
- Ventricular pacing
- Electrocardiogram artefact

V6 favours the diagnosis of VT in both LBBB-like and RBBB-like tachycardias. However, a triphasic QRS morphology in lead V1 indicates SVT with aberrancy.[7 8]

QRS activation delays

A significant delay during the *initial* part of the QRS complex is more compatible with VT (figure 4), whereas in SVT with aberrancy the QRS delay more frequently occurs in the *mid-to-terminal* portions of the QRS complex. Vereckei *et al* proposed that an index of slower conduction (assumed to be directly proportional to amplitude) at the initial versus terminal 40 ms portion of the QRS as evidenced by Vi/Vt ≤1 is suggestive of VT (figure 5).[9]

Vertical QRS axis

Extreme northwest axis (−90° to ±180°) is strongly suggestive of VT (figure 6).[10 11] Left-axis deviation in the context of a RBBB-like tachycardia or right-axis deviation in the context of a LBBB-like tachycardia also supports VT.[3 12] Vereckei *et al* reported that an initial R wave in aVR, indicative of inferior-to-superior ventricular activation, is sufficient to support the diagnosis of VT.[9] Of note, this feature was not observed in patients with pre-excited tachycardias. In the presence of a baseline SR ECG, Griffith *et al* found that a QRS axis change of equal or more than 40° during WCT is strongly predictive of VT.[13]

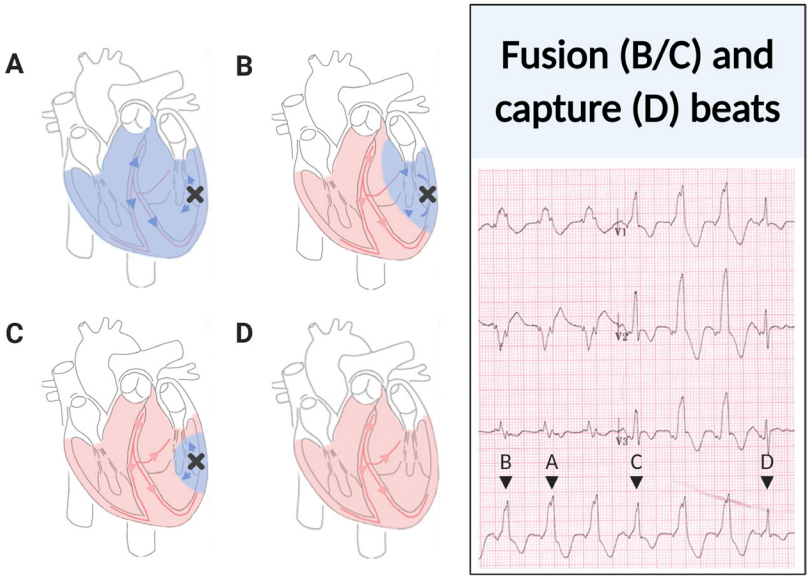

Figure 1 Generation of capture and fusion beats with corresponding changes on electrocardiogram due to competing activation from supraventricular and ventricular tachycardia source. Activation from a ventricular tachycardia source is indicated in *blue* and sinus rhythm is indicated in *red*.

Horizontal QRS axis

Positive concordance is defined as a positive QRS complex in all precordial leads. Negative concordance is defined as a negative QRS complex in all precordial leads. Strictly speaking, concordance is only present when all the precordial leads have monomorphic QRS complexes on the same side relative to the baseline. VT from the basal anterior left ventricle would be predicted to result in positive concordance while VT originating from the apical left or right ventricle result in negative concordance (figure 7).[7] Ventricular activation via the His-Purkinje system during SVT tends to produce an overall 'balanced' QRS complex from leads V1–6 as ventricular activation starts from the mid-inferior interventricular septum.

QRS duration

In the absence of pre-existing bundle branch block, QRS duration thresholds that may indicate VT are >140 ms for RBBB-like tachycardias and >160 ms for LBBB-like tachycardias.[3] The distinction between RBBB-like and LBBB-like tachycardias is based on the polarity of the QRS complex in lead V1 (RBBB-like has a positive polarity and LBBB-like has a negative polarity). Rarely, a narrower QRS is seen during tachycardia compared with SR. QRS narrowing in this context would support a diagnosis of VT.[5] This phenomenon is more commonly observed in patients with pre-existing bundle branch block who have VT originating from the left ventricular septum.

QRS morphology relative to baseline

The baseline ECG may identify pre-existing bundle branch block or pre-excitation. WCT with unchanged QRS configuration in leads I, II, (±III) and V1 is virtually synonymous with SVT, whereas a different QRS configuration in these leads is suggestive of VT.[14] Alternatively, an identical QRS vector during the initial 20 ms of WCT compared with SR favours SVT with aberrancy.[8] As discussed previously, differentiation between capture and fusion beats also rely on a comparison with baseline ECG.

LIMITATIONS OF ECG CRITERIA

While the criteria outlined previously are of value for distinguishing between VT and SVT, it is important to note that they are associated with limitations. Apart from AV dissociation, the majority of criteria are of limited value for distinguishing between VT and SVT with antegrade conduction via an accessory pathway. Individual criteria are also associated with specific limitations. While AV dissociation is associated with a high specificity in terms of diagnosing VT, definitive AV dissociation may only be appreciated in a minority

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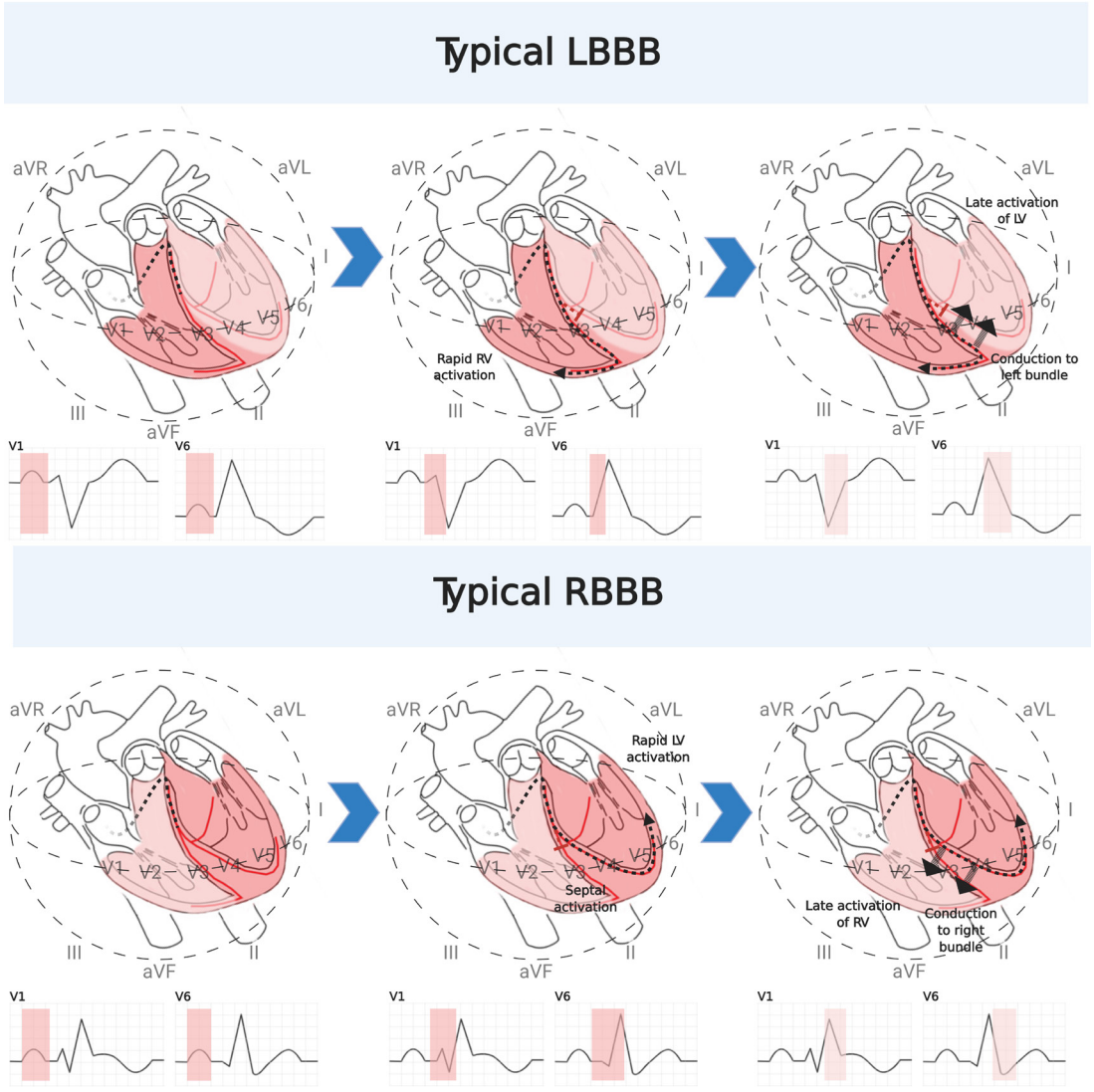

Figure 2 Activation patterns in typical right bundle branch block (RBBB) and left bundle branch block (LBBB) with corresponding changes on electrocardiogram. *Light red* areas are zones of late conduction while *dark red* areas indicate regions of early conduction.

of cases (overall sensitivity of 22%).[15] Furthermore, there are very rare exceptions of SVT which may demonstrate AV dissociation. In terms of analysis of ventricular activation, VT originating close to (or within) the proximal His-Purkinje system may result in early recruitment of the specialised conduction system producing ventricular activation patterns similar to SVT. Although positive or negative concordance in the precordial leads is a reliable ECG feature to distinguish between VT and SVT with aberrancy, an important caveat is that positive concordance may also be observed in SVT with antegrade AP conduction.[5] In isolation, the overall QRS duration is of limited value in distinguishing between VT and SVT, particularly if a patient has pre-existing bundle branch block.

EVOLUTION OF ELECTROCARDIOGRAPHIC ALGORITHMS

As discussed previously, the majority of criteria to distinguish between VT and SVT have limitations, which may result in diagnostic errors.[1 2 16] A number of algorithms have therefore been developed by incorporating several of the aforementioned as well as some additional criteria to increase the accuracy for diagnosing WCT (figure 8). Data on the accuracy of these algorithms (where available) are included in table 2. A brief summary of the algorithms is included below.

Wellens stepwise algorithm (1987)

The Wellens algorithm uses a series of high specificity but low sensitivity criteria in a stepwise fashion.[17] If a VT criterion is fulfilled at any stage, a diagnosis of VT can be made. Therefore, based on the Wellens criteria, SVT is a diagnosis of exclusion.

Kindwall combined algorithm (1988)

Using electrophysiological evaluation of LBBB-like tachycardia, Kindwall *et al* described four additional ECG features suggestive of VT.[6] Importantly, left-axis deviation in the presence of a LBBB-like tachycardia, which is a criterion in the Wellens algorithm, was not a useful marker for VT. The Kindwall

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Table 1 Likelihood ratio of ventricular tachycardia according to electrocardiographic features

Electrocardiographic features	Likelihood ratio of VT
QRS width >160 ms	22.9
Intrinsicoid deflection in lead V6 >80 ms	19.3
QRS axis	
Northwest (−90° to 180°)	7.9
Left-axis deviation with RBBB-like tachycardia	8.2
Right-axis deviation with LBBB-like tachycardia	3.9
RBBB-like pattern V1	
Taller left peak	50
Biphasic Rs or qR	4.0
LBBB-like pattern V1 or V2	
r≥40 ms	50
Notched S downstroke	50
Delayed S nadir >60 ms	50
V6 morphology	
Monophasic QS	50
Biphasic rS	50

Adapted from Lau *et al*.[35]
LBBB, left bundle branch block; RBBB, right bundle branch block; VT, ventricular tachycardia; WCT, wide complex tachycardia.

algorithm focuses on acquisition of simultaneous recordings in multiple leads and the importance of analysing both leads V1 and V2 to avoid misclassification, as the onset of QRS complex in lead V1 is often isoelectric. If all four criteria were present, the algorithm is diagnostic of VT with a sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and accuracy of 100%, 88.9%, 96.8%, 100% and 97.4%, respectively.

Brugada algorithm (1991)

Brugada *et al* refined the earlier algorithms in an attempt to create a simpler tool.[15] The authors introduced a novel criterion of absent RS complex in all precordial leads to be suggestive of VT (*ie, positive or negative concordance*). They also reported that an RS interval of >100 ms was not observed in any SVT and therefore was highly specific for VT. This finding was independent of the QRS duration and morphology. The authors reported that the algorithm is diagnostic of VT with a sensitivity, specificity, PPV, NPV and accuracy of 98.7%, 96.5%, 98.4%, 97.0% and 98.0%, respectively.[15]

Vereckei stepwise algorithm (2007)

Vereckei *et al* devised a simplified stepwise algorithm without the morphology criteria.[9] They also incorporated two new ECG features for VT: (1) initial R wave in aVR and (2) Vi/Vt ≤1. The algorithm was diagnostic of VT with a sensitivity, specificity, PPV, NPV and accuracy of 95.7%, 72.4%, 92.0%, 83.5% and 90.3%, respectively.

Vereckei aVR stepwise algorithm (2008)

The aVR algorithm is based on the concept of classifying VTs into two main categories based on the origin of ventricular activation and velocity of initial conduction.[18] In the Vereckei aVR algorithm, the criteria of AV dissociation was removed as it did not influence the overall accuracy. Nevertheless, the authors acknowledged that given the highly specific nature of this criteria, it may still be used preceding application of their algorithm.

'R'-wave peak time criteria (2010)

Pava *et al* analysed R-wave peak time (RWPT) in lead II, defined as QRS duration from the start of depolarisation until the first change of polarity, independent of whether the QRS deflection was positive or negative.[19] They reported a significantly longer RWPT in VT compared with SVT, with a cut-off of 50 ms being optimal for differentiating VT from SVT. This RWPT criterion was first reported to be diagnostic of VT with a sensitivity, specificity, PPV and NPV of 93.2%, 99.3%, 98.2% and 93.3%, respectively. However, subsequent studies have put into question the sensitivity and accuracy of this single, stand-alone criterion.[13 20]

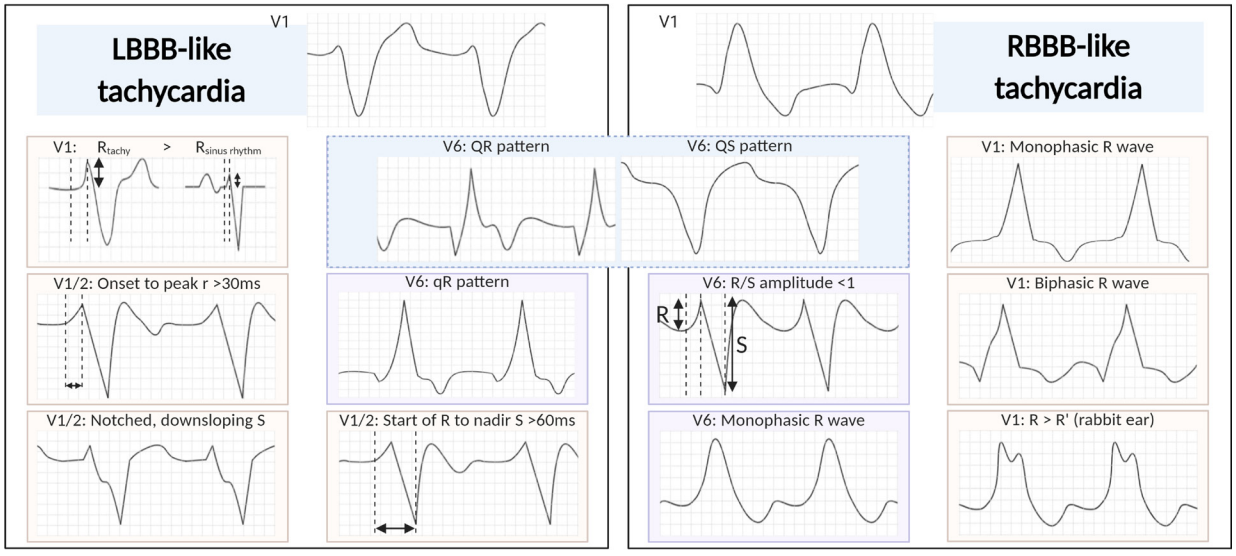

Figure 3 Atypical electrocardiographic features of right bundle branch block (RBBB) and left bundle branch block (LBBB) that are suggestive of ventricular tachycardia.

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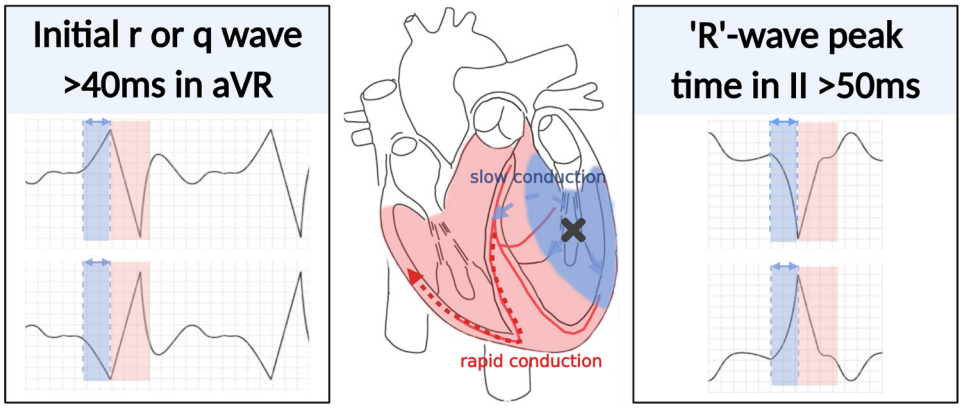

Figure 4 Features of slow initial ventricular conduction. *Light blue* arrows are myocyte-to-myocyte activation resulting in slow upstroke/downstroke on the corresponding electrocardiogram while *dark blue* arrows are engagement of the Purkinje system resulting in rapid ventricular activation with fast upstroke/downstroke on the corresponding electrocardiogram.

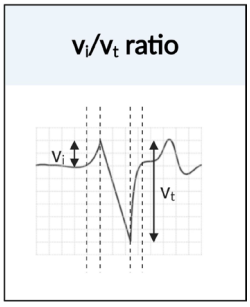

Figure 5 Vi/Vt measurement. Width of each square corresponds to 40 ms. The amplitude of the initial 40 ms of QRS activation is compared with the terminal 40 ms portion. A Vi/Vt ≤1 is suggestive of slow initial myocyte-to-myocyte conduction and therefore ventricular tachycardia.

Limb lead combined algorithm (2019)

It is important to emphasise that a major limitation of the described algorithms is the inability of independent authors to reproduce the reported sensitivity, specificity and accuracy in the original studies.[9 21–24] Chen *et al* proposed that this was related to the complexity of these algorithms secondary to the number of steps involved, and/or the difficulty and variability in the calculation of necessary measurements.[25] The limb lead algorithm was introduced as an alternative means for simple, accurate and rapid diagnosis of VT. The algorithm is based on the concept that unlike SVT with aberrancy which relies on horizontal conduction, most VTs propagate from the superior-to-inferior portions of the ventricle and may therefore be best detected in the frontal plane as assessed with the

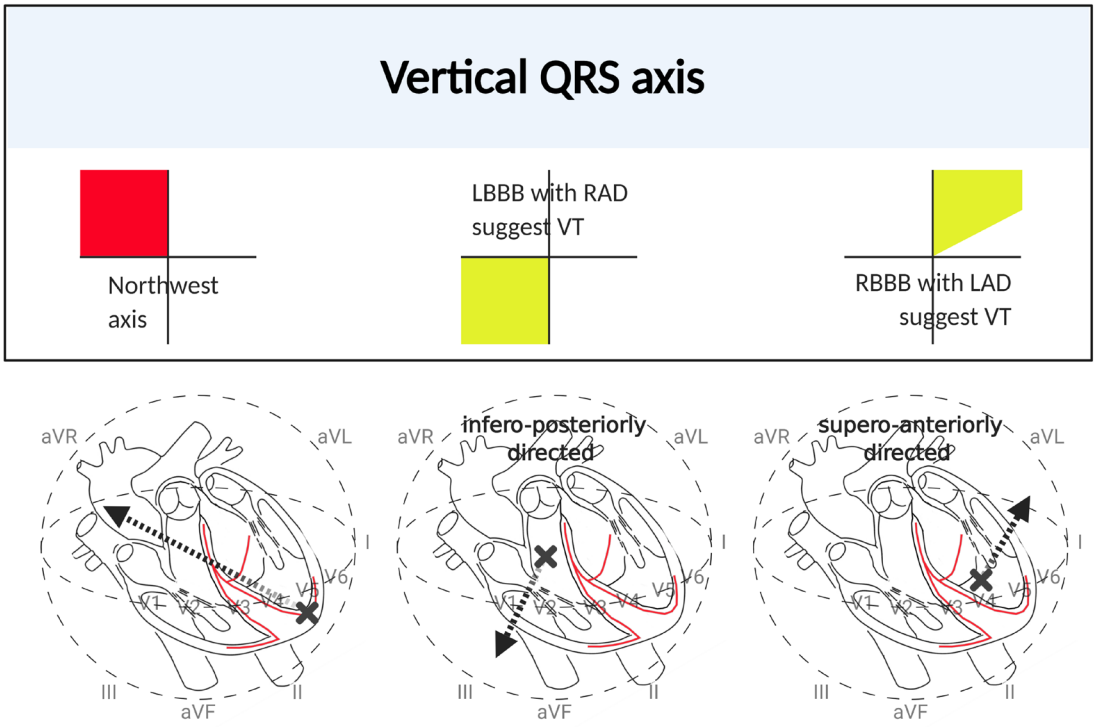

Figure 6 Features on vertical QRS axis suggestive of VT. Northwest axis (−90 to 180) in any context is strongly suggestive of VT. LBBB with RAD (+90 to 180) is suggestive of VT. RBBB with LAD (−30 to −90) is suggestive of VT. LAD, left axis deviation; LBBB, left bundle branch block; VT, ventricular tachycardia; RAD, right axis deviation; RBBB, right bundle branch block.

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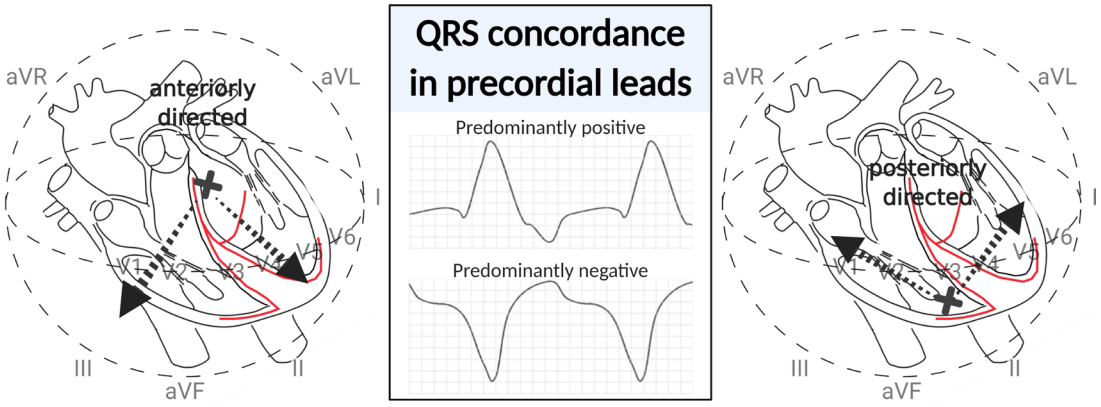

Figure 7 QRS concordance in precordial leads. Positive concordance is a predominantly positive QRS complex in V1–6 that occurs as a result of ventricular tachycardia originating from the basal anterior left ventricle. Negative concordance is a predominantly negative QRS complex in V1–6 as a result of ventricular tachycardia originating from the apical left or right ventricle. In contrast, ventricular activation via the His-Purkinje system during supraventricular tachycardia produces an overall 'balanced' QRS complex from leads V1–6 as it starts from the mid-inferior interventricular septum.

limb leads.[25] An advantage of this algorithm was that no measurements were required and hence it may be less prone to errors.

Scoring algorithm (2019)

Pachón *et al* devised a scoring algorithm for VT based on 7 of 16 criteria, each considered to have a high PPV.[26] Using this algorithm, the presence of a QRS morphology during WCT that is identical to the QRS on baseline ECG was strongly indicative of SVT (PPV 98%). In contrast, an abnormal Q wave on the baseline ECG, AV dissociation, presence of a Q wave or initial q in lead V6 with LBBB-like tachycardia, sudden normalisation and morphology changes in patients with AF on baseline ECG, WCT with complete or high-grade AV block, and contralateral BBB morphology in patients with organic BBB were each strongly associated with VT.

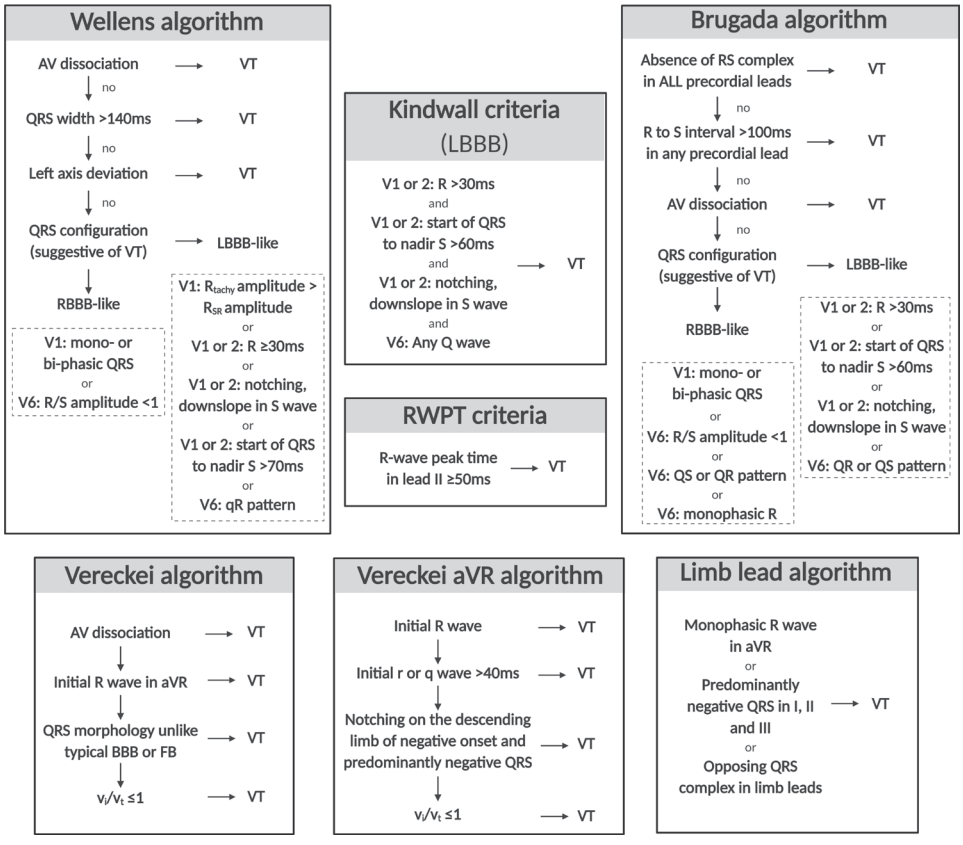
Wellens algorithm
AV dissociation → VT
no
QRS width >140ms → VT
no
Left axis deviation → VT
no
QRS configuration (suggestive of VT) → LBBB-like
RBBB-like
V1: mono- or bi-phasic QRS or V6: R/S amplitude <1
V1: Rtachy amplitude > RSR amplitude or V1 or 2: R ≥30ms or V1 or 2: notching, downslope in S wave or V1 or 2: start of QRS to nadir S >70ms or V6: qR pattern
Kindwall criteria (LBBB)
V1 or 2: R >30ms and V1 or 2: start of QRS to nadir S >60ms and V1 or 2: notching, downslope in S wave and V6: Any Q wave → VT
RWPT criteria
R-wave peak time in lead II ≥50ms → VT
Brugada algorithm
Absence of RS complex in ALL precordial leads → VT
no
R to S interval >100ms in any precordial lead → VT
no
AV dissociation → VT
no
QRS configuration (suggestive of VT) → LBBB-like
RBBB-like
V1: mono- or bi-phasic QRS or V6: R/S amplitude <1 or V6: QS or QR pattern or V6: monophasic R
V1 or 2: R >30ms or V1 or 2: start of QRS to nadir S >60ms or V1 or 2: notching, downslope in S wave or V6: QR or QS pattern
Vereckei algorithm
AV dissociation → VT
Initial R wave in aVR → VT
QRS morphology unlike typical BBB or FB → VT
vi/vt ≤1 → VT
Vereckei aVR algorithm
Initial R wave → VT
Initial r or q wave >40ms → VT
Notching on the descending limb of negative onset and predominantly negative QRS → VT
vi/vt ≤1 → VT
Limb lead algorithm
Monophasic R wave in aVR or Predominantly negative QRS in I, II and III or Opposing QRS complex in limb leads → VT

Figure 8 Wide complex tachycardia algorithms to distinguish between ventricular and supraventricular tachycardias. All the algorithms rely on specific features of ventricular tachycardia. If none are present, a diagnosis of supraventricular tachycardia is most likely. AV, atrioventricular; BBB, bundle branch block; FB, fascicular block; LBBB, left bundle branch block; RBBB, right bundle branch block; RWPT, R-wave peak time; VT, ventricular tachycardia.

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Table 2 Comparison of electrocardiographic algorithms in ventricular tachycardia

	Kindwall[6]	Brugada[15]	Vereckei[9]	Vereckei aVR[18]	'R'-wave peak time[19]	Limb lead[25]
Sensitivity (%)	100	98.7	95.7	96.5	93.2	87.2
Specificity (%)	88.9	96.5	72.4	75.0	99.3	90.8
PPV (%)	96.8	98.4	92.0	92.7	98.2	96.7
NPV (%)	100	97.0	83.5	86.6	93.3	70.0
Overall accuracy (%)	97.4	98.0	90.3	91.5	NA	88.1

*Note that these results have not been reproduced by independent authors.
NPV, negative predictive value; PPV, positive predictive value.

LIMITATIONS OF ELECTROCARDIOGRAPHIC ALGORITHMS

There are practical limitations to the application of conventional algorithms in clinical practice, especially while clinicians are under high-pressured situations. Moreover, the studies developing the algorithms have commonly involved invasive electrophysiolological studies and/or expert interpreters who were removed from the clinical care setting, and thus generalisability of the findings may be questionable.

In terms of limitations of specific algorithms, while the Wellens stepwise algorithm provides a systematic approach in the assessment of WCT, it has not been validated in independent studies and the overall accuracy remains questionable.[17] The Kindwall algorithm may be associated with difficulties in determining the interval from onset of QRS complex to nadir of the S wave.[6] Furthermore, the algorithm has not been tested in patients with an AP. The Brugada algorithm introduced several changes to the aforementioned algorithms while maintaining the morphological criteria. This contributed to a significant disadvantage in the Brugada algorithm as more than one-third of patients had discordance in their morphological criteria.[15] An important limitation of the Vereckei stepwise algorithm is that the third and fourth criteria were unable to reliably exclude SVT with antegrade AP conduction.[9] The Vereckei aVR stepwise algorithm was tested in a relatively small proportion of patients without structural heart disease and may therefore not apply to this cohort.[18] There may be difficulties in applying the RWPT criteria as the initiation and peak of ventricular complexes can be hard to define.[19] Furthermore, the Brugada and Vereckei aVR algorithms were demonstrated by independent authors to substantially underperform compared with the original studies.[21 23 27 28] Finally, the limb lead combined algorithm may misclassify VTs originating from the conduction system or intracavitary structures (eg, papillary muscles),[25] while the scoring algorithm has not been prospectively tested and used induced tachycardias in some patients.[26]

ALTERNATIVE DIAGNOSTIC METHODS

Despite improvements in ECG algorithms for the evaluation of WCT, there remain significant limitations with traditional models that rely on subjective interpretation and provide insufficient information regarding the probability of VT. Hence, alternative strategies have been developed.

VT score

Jastrzebski *et al* described a VT scoring system based on seven ECG features: initial R wave in lead V1; initial r >40 ms in lead V1/2; notched S wave in lead V1; initial R wave in aVR; R wave peak time ≥50 ms in lead II; no RS complex in leads V1–6; AV dissociation.[29] The model was highly specific (99.6%) for VT with a score of ≥3, despite the lack of sensitivity (56.9%). An advantage of this approach is that it considers a comprehensive list of ECG features of VT in each patient before proposing a diagnosis. Unlike previous methods, the VT score was designed to 'rule in' VT.

WCT formula

The WCT formula evaluates the WCT QRS duration and changes in amplitude (frontal and horizontal) during tachycardia compared with the baseline ECG.[30] By using a VT probability partition of 50%, this method produces a sensitivity, specificity, PPV, NPV and accuracy of 89.7%, 92.9%, 89.7%, 92.9% and 91.5%, respectively. An advantage of this approach was the ability to incorporate the formula into automated analysis algorithms, which may reduce inter-observer variability. Furthermore, it provides an estimation of the likelihood of VT. Kashou *et al* developed a new automated means to differentiate WCTs (WCT Formula II).[31] This was a system that was independent of the clinicians' ECG interpretation competency and, like the original WCT formula, could be integrated into contemporary ECG software. Of note, the WCT formula does however rely on pairing of WCT and baseline ECGs (*with QRS duration <120 ms and heart rate <100 bpm*), which may not always be possible.

VT prediction model

May *et al* also recently described an automated VT prediction model that used readily available ECG measurements to distinguish VT from SVT.[32] The model places more emphasis on the baseline ECG to arrive at the correct rhythm diagnosis. This system delivers an estimated probability of VT that may assist with the decision-making process. A benefit of the VT prediction model is that it does not have to be integrated into existing ECG interpretation softwares (unlike the WCT formula) but rather its use could be supported by mobile device applications or web-based platforms. However, further studies are still needed to refine this tool.

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Bayes approach

Independent studies of the various algorithms for differentiating VT and SVT have failed to reproduce results from the original studies. Overall, each algorithm has only been found to be modestly accurate for classification purposes.[24] Drew and Scheinman found that even in the best of circumstances, 1 in 10 WCT ECGs defy differentiation.[33] When using a Bayesian approach, an assumption of the likelihood of an event occurring is first made based on prior knowledge and subsequently modified as additional information is acquired.[34] Advantages of applying the Bayesian inference are that all relevant ECG features may be given due consideration (in contrast to the hierarchical nature of most algorithms which discards features lower in the diagnostic tree once a diagnosis is made) and it allows the problem of imperfect ascertainment of a particular ECG feature to be circumvented.[35] Adoption of the Bayesian approach in a study by Lau *et al* demonstrated significant improvement compared with the clinical assessment from three separate, independent cardiac specialists using the various algorithms.[35] However, the practical limitations of this approach include the use of multiple criteria and serial calculations. Furthermore, as each criterion may not be entirely independent, the final tabulated results may not accurately reflect the true likelihood of VT.

IMPORTANT CONSIDERATIONS AND PITFALLS IN A CLINICAL SITUATION

There are a number of important factors to consider when assessing a WCT in a clinical situation. First, clinical features are of limited value in distinguishing VT from SVT. While haemodynamic status may be a useful distinguishing marker,[16] it should be emphasised that a significant proportion of patients with VT may have no significant haemodynamic compromise.[36] In addition, some patients with SVT may present with haemodynamic compromise. Second, a pre-existing history of structural cardiac disease increases the pre-test probability of VT.[37] Of note however, the absence of these conditions has a poor negative predictive value.[37] Third, the presence of clinical or electrocardiographic features of VT are indicative of the condition, but their absence does not reliably exclude it. Fourth, the value of a baseline ECG should not be underestimated,[38] as it can be useful to determine capture and fusion beats, pre-existing bundle branch block, QRS axis change, QRS morphology change and underlying ischaemic heart disease. Finally, the features and algorithms described previously should be interpreted with caution in patients with anti-arrhythmic medications, severe electrolyte disturbance, medication-induced arrhythmia, known congenital heart disease or prior cardiac instrumentation (surgery or ablation).[39] Furthermore, its applicability in the immediate post-cardiac arrest or cardioversion stage remains limited.[39]

CONCLUSION

Accurate diagnosis of VT and SVT from an ECG requires a good understanding of the underlying principles of physiological cardiac activation and how this differs in specific pathologies. In this regard, numerous algorithms are available for assistance although each has its own inherent limitations and low reproducibility.

Key messages

In the context of a wide complex tachycardia:

- Based on the differences in ventricular activation patterns, a range of different electrocardiographic (ECG) criteria can be used to distinguish between ventricular tachycardia (VT) and supraventricular tachycardia with aberrant conduction.
- While some ECG criteria are strongly predictive of a diagnosis of VT, the majority of criteria are not diagnostic of VT in isolation.
- Various algorithms that combine multiple ECG criteria have been developed to improve the diagnostic value.
- While the existing diagnostic algorithms have been reported to enhance ECG-based diagnosis, reproducibility of diagnostic algorithms between studies has been limited and some algorithms do not lend themselves to rapid ECG diagnosis.

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Author note References which include a * are considered to be key references.

REFERENCES

1 Dancy M, Camm AJ, Ward D. Misdiagnosis of chronic recurrent ventricular tachycardia. *Lancet* 1985;2:320–3.
2 Stewart RB, Bardy GH, Greene HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med* 1986;104:766–71.
*3 Wellens HJ, Bär FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *Am J Med* 1978;64:27–33.
4 Issa Z, Miller JM, Zipes DP. *Clinical arrhythmology and electrophysiology: a companion to Braunwald's heart disease*. 3rd edn. Elsevier, 2018. https://books.google.co.uk/books?id=44MTtAEACAAJ

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*5 Wellens HJ. Electrophysiology: ventricular tachycardia: diagnosis of broad QRS complex tachycardia. *Heart* 2001;86:579–85.
6 Kindwall KE, Brown J, Josephson ME. Electrocardiographic criteria for ventricular tachycardia in wide complex left bundle branch block morphology tachycardias. *Am J Cardiol* 1988;61:1279–83.
7 Marriott HJ. Differential diagnosis of supraventricular and ventricular tachycardia. *Cardiology* 1990;77:209–20.
8 Sandler IA, Marriott HJ. The differential morphology of anomalous ventricular complexes of RBBB-type in lead V1. Ventricular ectopy versus aberration. *Circulation* 1965;31:551–6.
*9 Vereckei A, Duray G, Szénási G, *et al*. Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. *Eur Heart J* 2007;28:589–600.
10 Vereckei A. Current algorithms for the diagnosis of wide QRS complex tachycardias. *Curr Cardiol Rev* 2014;10:262–76.
11 Akhtar M, Shenasa M, Jazayeri M, *et al*. Wide QRS complex tachycardia. Reappraisal of a common clinical problem. *Ann Intern Med* 1988;109:905–12.
12 Swanick EJ, LaCamera F, Marriott HJ. Morphologic features of right ventricular ectopic beats. *Am J Cardiol* 1972;30:888–91.
13 Griffith MJ, de Belder MA, Linker NJ, *et al*. Multivariate analysis to simplify the differential diagnosis of broad complex tachycardia. *Br Heart J* 1991;66:166–74.
14 Dongas J, Lehmann MH, Mahmud R, *et al*. Value of preexisting bundle branch block in the electrocardiographic differentiation of supraventricular from ventricular origin of wide QRS tachycardia. *Am J Cardiol* 1985;55:717–21.
*15 Brugada P, Brugada J, Mont L, *et al*. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991;83:1649–59.
16 Morady F, Baerman JM, DiCarlo LA, *et al*. A prevalent misconception regarding wide-complex tachycardias. *JAMA* 1985;254:2790–2.
*17 Wellens HJ, Brugada P. Diagnosis of ventricular tachycardia from the 12-lead electrocardiogram. *Cardiol Clin* 1987;5:511–25.
*18 Vereckei A, Duray G, Szénási G, *et al*. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. *Heart Rhythm* 2008;5:89–98.
19 Pava LF, Perafán P, Badiel M, *et al*. R-wave peak time at DII: a new criterion for differentiating between wide complex QRS tachycardias. *Heart Rhythm* 2010;7:922–6.
20 Chen Q, Xu J, Gianni C, *et al*. Simple electrocardiographic criteria for rapid identification of wide QRS complex tachycardia: the new limb lead algorithm. *Heart Rhythm* 2020;17:431–8.
21 Jastrzebski M, Kukla P, Czarnecka D, *et al*. Comparison of five electrocardiographic methods for differentiation of wide QRS-complex tachycardias. *Europace* 2012;14:1165–71.
22 Szelényi Z, Duray G, Katona G, *et al*. Comparison of the "real-life" diagnostic value of two recently published electrocardiogram methods for the differential diagnosis of wide QRS complex tachycardias. *Acad Emerg Med* 2013;20:1121–30.
23 Isenhour JL, Craig S, Gibbs M, *et al*. Wide-complex tachycardia: continued evaluation of diagnostic criteria. *Acad Emerg Med* 2000;7:769–73.
24 Garner JB, Miller JM, Professor of Medicine and Director, Clinical Cardiac Electrophysiology and Training Program, Indiana University School of Medicine, Krannert Institute of Cardiology, 1800 N. Capitol Ave, Indianapolis, IN 46202, US. E: jmiller6@iu.edu. Wide complex tachycardia – ventricular tachycardia or not ventricular tachycardia, that remains the question. *Arrhythm Electrophysiol Rev* 2013;2:23–9.
25 Chen Q, Xu J, Gianni C, *et al*. Simple electrocardiographic criteria for rapid identification of wide QRS complex tachycardia: the new limb lead algorithm. *Heart Rhythm* 2020;17:431–8.
26 Pachón M, Arias MA, Salvador-Montañés Óscar, *et al*. A scoring algorithm for the accurate differential diagnosis of regular wide QRS complex tachycardia. *Pacing Clin Electrophysiol* 2019;42:625–33.
27 May AM, Brenes-Salazar JA, DeSimone CV, *et al*. Electrocardiogram algorithms used to differentiate wide complex tachycardias demonstrate diagnostic limitations when applied by non-cardiologists. *J Electrocardiol* 2018;51:110–9.
28 Kaiser E, Darrieux FCC, Barbosa SA, *et al*. Differential diagnosis of wide QRS tachycardias: comparison of two electrocardiographic algorithms. *Europace* 2015;17:1422–7.
29 Jastrzebski M, Sasaki K, Kukla P, *et al*. The ventricular tachycardia score: a novel approach to electrocardiographic diagnosis of ventricular tachycardia. *Europace* 2016;18:578–84.
30 May AM, DeSimone CV, Kashou AH, *et al*. The WCT formula: a novel algorithm designed to automatically differentiate wide-complex tachycardias. *J Electrocardiol* 2019;54:61–8.
31 Kashou AH, DeSimone CV, Deshmukh AJ, *et al*. The WCT formula II: an effective means to automatically differentiate wide complex tachycardias. *J Electrocardiol* 2020;61:121–9.
32 May AM, DeSimone CV, Kashou AH, *et al*. The VT prediction model: a simplified means to differentiate wide complex tachycardias. *J Cardiovasc Electrophysiol* 2020;31:185–95.
33 Drew BJ, Scheinman MM. ECG criteria to distinguish between aberrantly conducted supraventricular tachycardia and ventricular tachycardia: practical aspects for the immediate care setting. *Pacing Clin Electrophysiol* 1995;18:2194–208.
34 Bland JM, Altman DG. Bayesians and frequentists. *BMJ* 1998;317:1151–60.
35 Lau EW, Pathamanathan RK, Ng GA, *et al*. The Bayesian approach improves the electrocardiographic diagnosis of broad complex tachycardia. *Pacing Clin Electrophysiol* 2000;23:1519–26.
36 Morady F, Shen EN, Bhandari A, *et al*. Clinical symptoms in patients with sustained ventricular tachycardia. *West J Med* 1985;142:341–4.
37 Baerman JM, Morady F, DiCarlo LA, *et al*. Differentiation of ventricular tachycardia from supraventricular tachycardia with aberration: value of the clinical history. *Ann Emerg Med* 1987;16:40–3.
38 Kashou AH, Noseworthy PA, DeSimone CV, *et al*. Wide complex tachycardia differentiation: a reappraisal of the state-of-the-art. *J Am Heart Assoc* 2020;9:e016598.
39 Hollowell H, Mattu A, Perron AD, *et al*. Wide-complex tachycardia: beyond the traditional differential diagnosis of ventricular tachycardia vs supraventricular tachycardia with aberrant conduction. *Am J Emerg Med* 2005;23:876–89.

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